Inhalational Agents: Uptake

Uptake refers to transfer of inhalational anesthetic from the alveoli to the pulmonary circulation. However, uptake does not correlate with induction speed: only the partial pressure at the effect site (i.e. the brain and spinal cord) of inhaled anesthetics exerts an anesthetic effect. Dissolved anesthetic gas does not exert an effect. The partial pressure of anesthetic gas in the alveoli is thought to be equal to the partial pressure in the brain and spinal cord, therefore factors that increase uptake of anesthetic gas into the bloodstream, and thereby decrease the partial pressure of the gas in the alveoli, will decrease the partial pressure of anesthetic gas at the effect site. Generally, during induction, the more gas that is absorbed into the bloodstream (i.e. the greater the uptake), the slower the rise in F_A/F_i and the slower the induction.

Fresh gas flow: Strictly speaking this deals with delivery (not uptake) of the inhalational agent but the higher the fresh gas flow, the faster the F_i resembles the set points on the vaporizer and gas inlet mixer.

Alveolar minute ventilation: Again this technically involves inhalational agent delivery but the higher the alveolar MV, the faster F_A will approximate F_i and induction speed is increased. This effect is more noticeable with soluble gases as the absorbed gas is more rapidly replaced.

Solubility: More soluble anesthetics have increased uptake and therefore slower rises in F_A/F_i and slower inductions. Remember dissolved inhaled anesthetic is inactive inhaled anesthetic. Isoflurane is the only soluble inhaled anesthetic commonly used in the United States currently. Of dubious clinical importance, but lipidemia and hypothermia increase the solubility of inhaled anesthetics in blood while anemia and hyperthermia decrease it.

Cardiac Output: High cardiac output states quickly brings new blood to the alveoli and consequently a greater amount of inhaled anesthetic is able to be absorbed and dissolved. Therefore, increased cardiac output results in increased uptake and slowed induction.

Concentration effect: There's no way I can adequately cover this and stay under a page, but roughly when high concentrations of inhaled anesthetics are used, the partial pressure of anesthetic in the alveoli falls less than you'd intuit based on the degree of uptake. This causes a greater uptake of inhaled anesthetics the higher the concentration. This effect is greater with soluble inhaled anesthetics.

Second gas effect: Similar to concentration effect but this situation the absorption of a gas with a high concentration (nitrous or oxygen) results in increased uptake of a second gas (e.g. volatile anesthetic) since new fresh gas must replace the absorbed gas in the alveoli.

Left to right shunt: Increase in effective cardiac output is offset by increased mixed venous anesthetic concentration resulting in minimal change to uptake.

Right to left shunt: Large decrease in uptake due to decreased effective cardiac output or V/Q mismatch. This causes a more rapid rise in F_A/F_i but, uniquely, a slower induction because $P_A \neq P_a$ since pulmonary venous blood, fully saturated, mixes with the shunted blood that did not take up anesthetic. This effect is magnified for insoluble anesthetics.